WEST Search History

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DATE: Tuesday, March 09, 2004

Hide?	Set Name	Query	Hit Count
	DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ		
☐	L1	hiv! and iron! and chelator	821
☐	L2	hiv! and iron! near5 chelator	178
☐	L3	('20030158234')[URPN]	0
☐	L4	hiv! and iron! near5 chelator and (hydroxamate or deferoxamine)	54
	DB=EPAB; PLUR=YES; OP=ADJ		
☐	L5	WO-9939706-A1.did.	1
☐	L6	WO-9939706-A1.did.	1
☐	L7	hiv! and iron! near5 chelator and (hydroxypyridinon or deferiprone)	0
☐	L8	hiv! and iron! near5 chelator and (bleomycine)	0
	DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ		
☐	L9	hiv! and iron! near5 chelator and (hydroxypyridinon or deferiprone)	4
☐	L10	hiv! and iron! near5 chelator and (bleomycine)	0

END OF SEARCH HISTORY

L1 ANSWER 4 OF 7 MEDLINE on STN
TI Iron chelation decreases human immunodeficiency virus-1 Tat potentiated tumor necrosis factor-induced NF-kappa B activation in Jurkat cells.
SO European cytokine network, (1997 Mar) 8 (1) 37-43.
Journal code: 9100879. ISSN: 1148-5493.
AU Shatrov V A; Boelaert J R; Chouaib S; Droge W; Lehmann V
AB TNF-alpha stimulates HIV-1 replication via activation of the transcription factor NF-kappa B. TNF-mediated activation of NF-kappa B is known to involve the intracellular formation of reactive oxygen intermediates (ROIs). We recently demonstrated that HIV-1 Tat protein potentiates TNF-induced NF-kappa B activation by downregulation of manganese-dependent superoxide dismutase (MnSOD), shifting the cellular redox state towards pro-oxidative conditions. This study shows that treatment of Jurkat cells with iron chelator deferoxamine (DFO) strongly decreases HIV-1 Tat-potentiated TNF-induced NF-kappa B activation but does not modify NF-kappa B activation by TNF-alpha. The ability of iron chelators to reduce Tat-potentiated TNF-induced NF-kappa B binding activity suggests that iron and intracellular hydroxyl radicals (OH.) are required for Tat effect. Moreover, we have shown that exogenously generated OH. markedly enhanced TNF-induced NF-kappa B activation in a dose-dependent manner while was not sufficient to trigger activation of NF-kappa B by itself. In addition, iron chelators had no effect either on MnSOD activity or on the decrease of this activity by Tat. Iron chelators had also no effect on the ratio of reduced glutathione (GSH) to oxidized glutathione (GSSG), but could elevate the GSH:GSSG ratio decreased by Tat protein. These observations suggest that the formation of intracellular OH. in the presence of iron ions play a major role in HIV-1 Tat enhancement of TNF-induced NF-kappa B activation and that iron chelation may protect Jurkat T cells, at least in part, against oxidative stress induced by Tat.

L1 ANSWER 5 OF 7 MEDLINE on STN
TI Altered iron metabolism in HIV infection: mechanisms, possible consequences, and proposals for management.
SO Infectious agents and disease, (1996 Jan) 5 (1) 36-46. Ref: 99
Journal code: 9209834. ISSN: 1056-2044.
AU Boelaert J R; Weinberg G A; Weinberg E D
AB The progression of human immunodeficiency virus (HIV) infection toward its more advanced stages is accompanied by increasing body iron stores. Iron accumulates in macrophages, microglia, endothelial cells, and myocytes. The iron burden is especially heavy in bone marrow, brain white matter, muscle, and liver. Excess iron potentially enhances oxidative stress, impairs several already compromised immune defense

mechanisms, and directly promotes the growth of microbial cells. Thus, we hypothesize that the prevention (or at least, reduction) of iron loading might slow the progression of the infectious complications of HIV infection, and perhaps indirectly, the HIV infection itself. A twofold strategy is proposed, consisting of (a) limitation of iron intake through the alimentary, parenteral, and respiratory routes, and (b) possibly the use of iron chelator drugs that could decrease the iron burden, redistribute the metal to the erythroblasts, and suppress the growth of microorganisms. This approach is still to be considered as hypothetical. However, the available data suggest that there is an urgent need for careful clinical studies to clarify the role of iron status on the course of HIV infection.

(FILE 'HOME' ENTERED AT 10:21:20 ON 09 MAR 2004)

FILE 'MEDLINE' ENTERED AT 10:22:19 ON 09 MAR 2004

L1	7	S	HIV AND (IRON CHELATOR)
		E	ASBECK/AU
		E	MARX J/AU
L2	135	S	E12
L3	5	S	L2 AND CHELATOR
L4	0	S	BLEOMYCINE AND CHELATOR

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 169. Document ID: US 5605810 A

L2: Entry 169 of 178 File: USPT Feb 25, 1997

US-PAT-NO: 5605810
DOCUMENT-IDENTIFIER: US 5605810 A
** See image for Certificate of Correction **

TITLE: NADH oxidase assay for neoplasia determination

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 170. Document ID: US 5403834 A

L2: Entry 170 of 178 File: USPT Apr 4, 1995

US-PAT-NO: 5403834
DOCUMENT-IDENTIFIER: US 5403834 A

TITLE: Synthetic catalytic free radical scavengers useful as antioxidants for prevention and therapy of disease

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 171. Document ID: US 5380747 A

L2: Entry 171 of 178 File: USPT Jan 10, 1995

US-PAT-NO: 5380747
DOCUMENT-IDENTIFIER: US 5380747 A

TITLE: Treatment for atherosclerosis and other cardiovascular and inflammatory diseases

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

→ ☑ 172. Document ID: EP 1257261 A2, WO 200112168 A2, NL 1012825 C2, AU 200064831 A

L2: Entry 172 of 178 File: DWPI Nov 20, 2002

DERWENT-ACC-NO: 2001-211128
DERWENT-WEEK: 200301
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TITLE: New product for treating viral infections, particularly HIV infection, comprising an iron chelator and a component comprising another virus-inhibiting compound

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 173. Document ID: EP 1006112 A1

L2: Entry 173 of 178 File: DWPI Jun 7, 2000

Hit List

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Search Results - Record(s) 1 through 1 of 1 returned.

☐ 1. Document ID: WO 9939706 A1

L5: Entry 1 of 1 File: EPAB Aug 12, 1999

PUB-NO: WO0009939706A1
DOCUMENT-IDENTIFIER: WO 9939706 A1
TITLE: PHARMACEUTICALS COMPRISING N,N'-BIS(2- HYDROXYBENZYL) ETHYLENEDIAMINE-N, N'-DIACETIC ACID FOR IRON CHELATING THERAPY

PUBN-DATE: August 12, 1999

INVENTOR-INFORMATION:

NAME	COUNTRY
BERGERON, RAYMOND J JR	US

ASSIGNEE-INFORMATION:

NAME	COUNTRY
UNIV FLORIDA	US
BERGERON RAYMOND J JR	US

APPL-NO: US09902388
APPL-DATE: February 3, 1999

PRIORITY-DATA: US07360398P (February 4, 1998)

INT-CL (IPC): A61 K 31/195; A61 K 9/08
EUR-CL (EPC): A61K031/198

ABSTRACT:

CHG DATE=19990902 STATUS=O>The use of N,N'-bis(2- hydroxybenzyl) ethylenediamine-N, N'-diacetic acid (HBED) in iron chelating therapy is disclosed. In particular, the invention relates to the subcutaneous use of HBED for treating mammals with a disease treatable by an iron chelator such as iron overload, especially transfusional iron overload.

Full | Title | Citation | Front | Review | Classification | Date | Reference | [illegible] | Claims | KWIC | Drawi Desc | Image

Clear | Generate Collection | Print | Fwd Refs | Bkwd Refs | Generate OACS

Term	Documents
WO-9939706-A1	1
WO-9939706-A1S	0
WO-9939706-A1.DID..EPAB.	1
(WO-9939706-A1.DID.).EPAB.	1